This listing of claims will replace all prior versions, and listings, of claims in the application:

Listing of Claims:

Claims 1-49 (canceled)

Claim 50 (currently amended): A method for inducing formation of new blood vessels in a mammal having chronic or acute ischemia, wherein the method comprises administering to the mammal an effective amount of a vascular endothelial growth factor (VEGF) or a hematopoietic factor sufficient to form the new blood vessels in the mammal, and increasing endothelial progenitor cell (EPC) frequency by at least about 20% as determined by a standard EPC isolation assay, wherein the hematopoietic factor is a granulocyte-macrophage colony stimulating factor (GM-CSF), stem cell factor (SCF), stromal cell-derived factor (SDF-1), granulocyte-colony stimulating factor (G-CSF), monocyte-colony stimulating factor (M-CSF), angiopoietin-1, angiopoietin-2, fetal liver tyrosine kinase 3 (FLT-3) ligand, or an effective fragment thereof, wherein the mammal is a rodent or a primate, provided that when VEGF is administered, the method further comprises administering at least one hematopoietic factor or an effective fragement thereof to the mammal.

Claim 51 (canceled)

Claim 52 (previously presented). The method of claim 50, wherein the factor is GM-CSF, and amount of the GM-CSF administered to the mammal is sufficient to increase frequency of endothelial progenitor cells (EPC) in the mammal.

Claims 53-54 (canceled)

Claim 55 (previously presented). The method of claim 50, wherein the amount of factor administered to the mammal is sufficient to increase blood vessel length in the mammal.

Claim 56 (previously presented) The method of claim 55, wherein the increase in blood vessel length is at least about 5% as determined by a standard blood vessel length assay.

Claim 57 (previously presented). The method of claim 53, wherein the amount of factor administered to the mammal is further sufficient to increase blood vessel diameter in the mammal.

Claim 58 (previously presented) The method of claim 56, wherein the increase in blood vessel diameter is at least about 5% as determined by a standard blood vessel diameter assay.

Claim 59 (previously presented). The method of claim 50, wherein the amount of factor administered to the mammal is sufficient to increase EPC differentiation following tissue ischemia.

Claim 60 (previously presented) The method of claim 59, wherein the increase in EPC differentiation is at least about 20% as determined by a standard hindlimb ischemia assay.

Claim 61 (previously presented). The method of claim 50, wherein the amount of administered factor is sufficient to increase neovascularization by at least about 5% as determined by a standard cornea micropocket assay.

Claim 62 (previously presented). The method of claim 50, wherein the amount of administered factor is sufficient to increase EPC incorporation into foci.

Claim 63 (previously presented) The method of claim 62, wherein the increase in EPC incorporation into foci is at least about 20% as determined by a standard rodent bone marrow (BM) transplantation model.

Claim 64 (canceled)

Claims 65 (previously presented) The method of claim 63, wherein the mammal has ischemic tissue which comprises tissue from a limb, graft, or organ.

Claim 66 (previously presented) The method of claim 65, wherein the tissue is associated with the circulatory system or the central nervous system.

Claim 67. (previously presented) The method of claim 65, wherein the tissue is heart or brain tissue.

Claim 68 (previously presented). The method of claim 50, wherein the factor is co-administered with at least one angiogenic protein.

Claim 69 (canceled)

Claim 70. (currently amended) The method of claim 68, wherein the angiogenic protein is acidic fibroblast growth factor (aFGF), ~~basic fibroblast growth factor (bFGF), vascular endothelial growth factor (VEGF-1),~~ epidermal growth factor (EGF), transforming growth factor a and (3 (TGF-a and TFG-P), platelet-derived endothelial growth factor (PD-ECGF), platelet-derived growth factor (PDGF), tumor necrosis factor a (TNF-a), hepatocyte growth factor (HGF), insulin like growth factor (IGF), erythropoietin, colony stimulating factor (CSF), macrophage-CSF (M-CSF), angiopoetin-1 (Angl) or nitric oxidesynthase (NOS); or a fragment thereof.

Claim 71 (canceled)

Claims 72 (previously presented) A method for preventing or reducing the severity of blood vessel damage in a mammal having chronic or acute ischemia, wherein the method comprises administering to the mammal an effective amount of granulocyte macrophage-colony stimulating factor (GM-CSF); and exposing the mammal having the chronic or acute ischemia to conditions conducive to damaging the blood vessels, the amount of GM-CSF being sufficient to prevent or reduce the severity of the blood vessel damage in the mammal.

Claim 73. (previously presented) The method of claim 72, wherein the conditions conducive to the blood vessel damage are an invasive manipulation or ischemia.

Claim 74. (previously presented) The method of claim 73, wherein the invasive manipulation is surgery.

Claim 75. (previously presented) The method of claim 73, wherein the ischemic is associated with at least one of infection, trauma, graft rejection, cerebrovascular ischemia, renal ischemia, pulmonary ischemia, limb ischemia, ischemic cardiomyopathy, or myocardial ischemia.

Claim 76. (previously presented) The method of claim 72, wherein the GM-CSF is administered to the mammal at least about 12 hours before exposing the mammal to the conditions conducive to damaging the blood vessels.

Claim 77. (previously presented) The method of claim 76, wherein the GM-CSF is administered to the mammal between from about 1 to 10 days before exposing the mammal to the conditions conducive to damaging the blood vessels.

Claim 78. (previously presented) The method of claim 76, wherein the method further comprises administering the GM-CSF to the mammal following the exposure to the conditions conducive to damaging the blood vessels.

Claim 79. (previously presented) A method for enhancing endothelial progenitor cell (EPC) mobilization in a mammal having chronic or acute ischemia, wherein the method comprises administering an effective amount of at least one hematopoietic factor sufficient to enhance the EPC mobilization in the mammal having the chronic or acute ischemia.

Claims 80-81 (canceled)

Claim 82 (currently amended) A method for inducing formation of new blood vessels in a mammal having chronic or acute ischemia, and increasing endothelial progenitor cell (EPC) frequency, wherein the method comprises administering to the mammal an effective amount of a ~~vascular endothelial growth factor (VEGF) or a~~ hematopoietic factor sufficient to form the new blood vessels in the mammal, wherein the hematopoietic factor is a granulocyte-macrophage colony stimulating factor (GM-CSF), stem cell factor (SCF), stromal cell-derived factor (SDF-1), granulocyte-colony stimulating factor (G-CSF), monocyte-colony stimulating factor (M-CSF), angiopoietin-1, angiopoietin-2, fetal liver tyrosine kinase 3 (FLT-3) ligand, or an effective fragment thereof, wherein the mammal is a rodent or a primate.

Claim 83 (previously presented) The method of claim 82, wherein the progenitor cell (EPC) frequency is increased by at least about 20% as determined by a standard EPC isolation assay.

Claim 85 (new) A method for inducing formation of new blood vessels in a mammal having chronic or acute ischemia, wherein the method comprises administering to the mammal an effective amount of a vascular endothelial growth factor (VEGF) or a hematopoietic factor sufficient to form the new blood vessels in the mammal, and increasing endothelial progenitor cell (EPC) frequency by at least about 20% as determined by a standard EPC isolation assay, wherein the hematopoietic factor is a granulocyte-macrophage colony stimulating factor (GM-CSF), stem cell factor (SCF), stromal cell-derived factor (SDF-1), granulocyte-colony stimulating factor (G-CSF), monocyte-colony stimulating factor (M-CSF), angiopoietin-1, angiopoietin-2, fetal liver tyrosine kinase 3 (FLT-3) ligand, or an effective fragment thereof, wherein the mammal is a rodent or a primate.